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LETTERS TO THE EDITOR

Long-term evolution of cardiomyopathy in dialysis patients

To the Editor: I read with great interest the recent article that appeared in *Kidney International* by Foley and co-workers [1]. Their study is quite novel because of the number of echocardiograms performed in sequence during a relatively long period of follow-up. However, I would like to make a few comments on the study:

First, blood pressure was defined as the average of all measurements done. It might have been more appropriate to analyze pre- and post-dialysis blood pressure levels separately, since left ventricular mass (LVM) index has been shown previously to correlate better with pre-dialysis systolic blood pressure level [2, 3].

Second, it is not clear whether the patients' antihypertensive medications were modified during the study period. If angiotensin-converting enzyme inhibitors were started after the first year, this would explain, at least in some patients, why further significant LVM enlargement did not develop [4].

Finally, I do not share the authors' conclusion that intervention beyond the first year of dialysis treatment might be relatively ineffective to prevent progressive cardiac enlargement. In contrast, I think that the relatively normal blood pressure levels and desirable hemoglobin levels achieved prevented progression beyond one year. Indeed, previous studies demonstrated a significant regression in LVM index after partial correction of anemia with erythropoietin [5]. The problem remaining to be solved is why a significant cardiac enlargement developed during the first year of the study, in spite of blood pressure and hemoglobin levels comparable with those of the later periods.

In conclusion, much effort should be made to prevent currently known risk factors from developing during the entire course of the dialysis therapy, though further studies are needed to find out the exact mechanisms responsible for the progressive cardiac enlargement.

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REFERENCES

1. FOLEY RN, PARFREY PS, KENT GM, HARNETT JD, MURRAY DC, BARRE PE: Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 54:1720–1725, 1998
2. ERTÜRK S, ERTUG AE, ATES K, DUMAN N, ASLAN SM, NERGISOGLU G, DIKER E, EROL C, KARATAN O, ERBAY B: Relationship of ambulatory blood pressure monitoring data to echocardiographic findings in haemodialysis patients. *Nephrol Dial Transplant* 11:2050–2054, 1996
3. CONLON PJ, WALSHE JJ, HEINLE SK, MINDA S, KRUCOFF M, SCHWAB SJ: Predialysis systolic blood pressure correlates strongly with mean 24-hour systolic blood pressure and left ventricular mass in stable hemodialysis patients. *J Am Soc Nephrol* 7:2658–2663, 1996
4. CANNELLA G, PAOLETTI E, DELFINO R, PELOSO G, ROLLA D, MOLINARI S: Prolonged therapy with ACE inhibitors induces a regression of left ventricular hypertrophy of dialyzed uremic patients independently from hypotensive effects. *Am J Kidney Dis* 30:659–664, 1997
5. PASCUAL J, TERUEL JL, MOYA JL, LIANO F, JIMENEZ-MENA M, ORTUNO J: Regression of left ventricular hypertrophy after partial correction of anemia with erythropoietin in patients on hemodialysis: a prospective study. *Clin Nephrol* 35:280–287, 1991

To the Editor: The question of whether cardiac function improves or worsens after initiation of dialysis therapy is still open. The paper by Foley et al [1] is a step forward towards resolving this issue. The authors concluded that left ventricular dilation with compensatory hypertrophy continued after the start of dialysis therapy, particularly in the first year of therapy. A few relevant papers concerning this problem were published earlier, but the Authors did not cite them [2, 3].

The study by Foley et al was prospective. The patient subset was highly selected: only 29 of 433 patients who started the study were analyzed. The patient subset was older than the parent population—58 vs. 51 years. Age is one of the major risk factors for the development of congestive heart failure in dialysis patients. The prevalence of congestive heart failure was 21% at the start of the study. The number of patients who developed congestive heart failure during the 41-month follow-up is not given. Because cardiovascular response differs among hemodialysis (HD) patients and peritoneal dialysis patients [4], we believe that echocardiographic findings in these two groups should be analyzed separately.

It is very likely that hyperparathyroidism contributes to left ventricular hypertrophy (LVH) in dialysis patients and affects cardiac function [4, 5]. There are no data in the paper on PTH or alkaline phosphatase; in our opinion, normal plasma calcium and phosphate concentrations do not exclude hyperparathyroidism. If the authors look at the risk factors for the progression of cardiomegaly in dialysis patients, the parameters describing parathyroid function must be taken into account. Because parathyroid function fluctuates as dialysis is continued and PTH concentration varies irrespective of the dura-

tion of renal replacement therapy, cardiac performance may change as a function of PTH level but not as the effect of duration of therapy. Such a possibility is suggested by the data presented in Table 3. Results given in the second and third columns show that cardiac mass, volume and function indexes deteriorate in some patients but improve or do not change in others. The differences may depend on mode of dialysis and PTH concentration.

Therapy with EPO leads to the regression of LVH in dialysis patients. The mean hemoglobin level in the study group presented in the first column of Table 4 is 8.6 g/dl (range, 8.1–9.2), but the data plotted in Figure 1 reveal that as many as 9 HD patients had a hemoglobin level below 8 g/dl. We are curious as to whether these patients, particularly those with hemoglobin levels of 6–7 g/dl, were given EPO. If yes, it should be mentioned, because this hormone affects cardiac function regardless of renal replacement therapy. If EPO was not given to these patients, one wonders why the authors did not address the severe anemia.

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REFERENCES

1. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE: Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 54:1720–1725, 1998
2. Cohen JL, Barooah B, Segal KR, Batuman V: Two-dimensional echocardiographic findings in patients on hemodialysis for more than six months. *Am J Cardiol* 60:743–745, 1987
3. Leenen FH, Smith DL, Khanna R, Oreopoulos DG: Changes in left ventricular hypertrophy and function in hypertensive patients on continuous ambulatory peritoneal dialysis. *Am Heart J* 110:102–106, 1985
4. Harnett JD, Parfrey PS, Griffiths SM, Gault MH, Barre P, Guttmann RD: Left ventricular hypertrophy in end-stage renal disease. *Nephron* 48:107–115, 1988
5. Hara S, Ubara Y, Arizono K, Ikeguchi H, Katori H, Yamada A, Ogura Y, Murata H, Mimura N: Relation between parathyroid hormone and cardiac function in long-term hemodialysis patients. *Miner Electrolyte Matab* 21:72–76, 1995

To the Editor: We would like to comment on the study of Foley et al [1]. In our opinion it is not correct to speak about the "natural history of cardiomyopathy" because the development of this cardiomyopathy is highly dependent on the way patients are treated, particularly the extent to which the treating team succeeds in controlling "volume."

The fatalistic attitude that unfortunately prevails in many dialysis centres is supported by the conclusion that "progressive left ventricular dilatation with compensatory hypertrophy is the major long-time evolutionary pattern" and that "intervention beyond one year may be relatively ineffective."

The fact that better results were obtained with peritoneal dialysis may simply be due to better volume control in those patients. Other studies [2] have shown that this is not always the case. The absence of a relationship with blood pressure could be related to the confounding effect of antihypertensive drugs, but probably also to an independent effect of volume retention.

We recently showed [3] that prolonged strict volume control can cause regression of dilatation and hypertrophy of the left ventricle while abolishing the need for antihypertensive drugs. An extreme example of a patient in whom volume control caused complete regression of severe cardiomyopathy without hypertension [4] illustrates the importance of hypervolemia as an independent risk factor. Clearly (as Foley et al stressed in earlier publications) only intervention studies can definitely resolve the complicated problems of dialysis morbidity.

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REFERENCES

1. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE: Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 54:1720–1725, 1998
2. Lameire N, Vanholder RC, Van Loo A, Lambert MC, Vijt D, Van Bockstaele, Vogeleere P, Ringoir SM: Cardiovascular diseases in peritoneal dialysis patients: the size of the problem. *Kidney Int Suppl* 56:S28–S36, 1996
3. Özkahya M, Ok E, Cirit M, Aydin Ş, Akçiçek F, Başçi A, Dorhout Mees EJ: Regression of left ventricular hypertrophy in haemodialysis patients by ultrafiltration and reduced salt intake without antihypertensive drugs. *Nephrol Dial Transplant* 13:1489–1493, 1998
4. Töz H, Özerkan F, Ünsal A, Soyda C, Dorhout Mees EJ: Dilated uremic cardiomyopathy in a dialysis patient cured by persistent ultrafiltration. *Am J Kidney Dis* 32:664–668, 1998

Reply from the authors

To the Editor: We would like to thank all the correspondents for their interest and helpful queries and comments.

Dr. Erturk makes several reasonable points. It certainly would have been of interest to look at the potential role of post-dialysis blood pressures in the hemodialysis patients. The short answer is that we did not record them, partly reflecting the fact that the study began in the early 1980s. It is hardly fair to say that post-dialysis blood pressure is a better *predictor* of LV mass index than pre-dialysis blood pressure. We have previously shown, in a prospective cohort study of over a thousand